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PESTS NOT KNOWN TO OCCUR IN THE UNITED STATES OR OF LIMITED DISTRIBUTION NO. 89: SOYBEAN DWARF VIRUS

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Disease

Soybean dwarf, subterranean clover red leaf

Pathogen

Soybean dwarf virus (SDV)

Synonym

Subterranean clover red leaf virus (SCRLV) (Ashby and Johnstone 1985)

Group

Luteovirus. Cryptogram R/1:1.9/30:S/S:S/Ap (A. D. Hewings, pers. comm.).

Economic Importance

Soybean dwarf virus is considered one of three very important soybean viruses in Japan (Tamada 1977). The percentage of infected soybean plants closely correlates with the percentage of yield loss. A 50-percent infection resulted in 40-percent yield loss. The number of pods are reduced, decreasing the weight of seeds produced per plant (Banba et al. 1986, Tamada 1975).

SDV is also one of the two most damaging viruses to pasture and vegetable legumes in temperate Australia. Heavy losses occur in annual legume crops in New Zealand (Ashby and Johnstone 1985). Beans suffer a serious yellowing disease in New Zealand (Ashby 1980, Ashby et al. 1979), including Japan (Murayama et al. 1975). Yield for SDV-inoculated beans was reduced 77 percent (Ashby et al. 1979). Heavy losses for broad beans (Ashby et al. 1979, Johnstone 1978) and subterranean clover (Johnstone and McLean 1987) have been reported in Australia and New Zealand. Peas in Tasmania are commonly infected but usually escape severe loss because tolerance to SDV is high in pea cultivars there. Early, natural infection of peas, however, resulted in stunting and severe fungal infections, often ending in plant death (Ashby et al. 1979, Johnstone 1978).

SDV has the potential to be a serious pest if it became established in the United States. It is a temperate-region virus, its hosts are important crops, its vector occurs in U.S. legume crop areas, the vector feeds on many crops and weeds, losses are heavy in foreign countries, several legumes are symptomless hosts, and soybean resistance is limited (Damsteegt and Hewings 1986).

General Distribution

Australia (New South Wales, South Australia, Tasmania, Victoria, Western Australia (Johnstone, Duffus et al. 1982)); Japan (Hokkaido and northern Honshu (Banba et al. 1986)); New Zealand (North and South Islands (Ashby et al. 1979)).

Soybean dwarf virus distribution map.

An SDV-like isolate in central California, United States, appears identical to the Australian isolate of SDV in particle morphology, serological tests, and symptomatically but is transmitted by pea aphid, but not by foxglove aphid (Johnstone, Liu, Duffus 1984). Recent information suggests that an SDV-like isolate occurs in subterranean clover in the United States. It is also transmitted only by pea aphid (V. D. Damsteegt, pers. comm.).

Studies show that the soybean dwarf disease reported elsewhere is incited by other viruses: Indonesian soybean dwarf virus in Indonesia (Iwaki et al. 1980), and African soybean dwarf virus in Nigeria (Rossel 1985, Rossel and Thottapilly 1982, Rossel et al. 1984).

Hosts

Mostly in Fabaceae. Naturally infected hosts include the following species, with an asterisk for those species showing no symptoms: Beta vulgaris (sugar beet), Calandrinia ciliata (= C. caulescens) (redmaids), Erodium moschatum (whitefilaree)*

(Ashby et al. 1979, Johnstone and Duffus 1984), Glycine max (soybean) (Tamada 1970), Gomphrena globosa (globe amaranth) (Johnstone and Duffus 1984), Lens culinaris (= L. esculenta) (lentil), Lupinus albus (white lupine), Lupinus angustifolius (European blue lupine), Lupinus cosentini, Lupinus luteus (European yellow lupine)* (Ashby et al. 1979), Medicago polymorpha (California burclover) (Johnstone and Duffus 1984), Phaseolus vulgaris (bean), Pisum sativum (pea) (Tamada 1973), Rumex obtusifolius (broadleaf dock)*, Trifolium dubium (small hop clover), T. fragiferum (strawberry clover)*, T. hybridum (alsike clover) (Ashby et al. 1979), T. pratense (red clover)*, T. repens (white clover)* (Tamada 1973), T. striatum (striate clover)*, T. subterraneum (subterranean clover) (Ashby et al. 1979), Vicia articulata (single-flowered vetch) (Johnstone and Duffus 1984), V. faba (broad bean) (Wilson and Close 1973), and V. sativa (vetch) (Ashby et al. 1979).

Characters

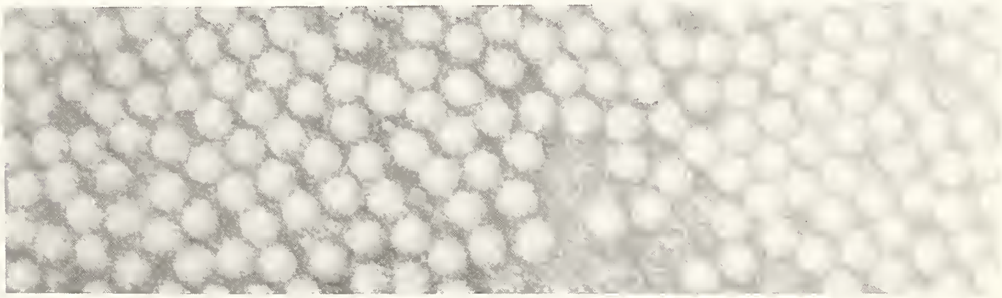
SDV is identified by particle morphology, host symptomatology, vector specificity, and serological tests.

Particles isometric (Fig. 1), diameter about 25 nm, with some fine surface structure (Hewings et al. 1986, Kojima and Tamada 1976).

Transmission - By grafts and aphid vectors. No transmission through sap inoculation, seed (Tamada et al. 1969), or vector progeny (Tamada 1975).

The major aphid vector is Acyrthosiphon (= Aulacorthum) solani (Kaltenbach), foxglove aphid. Other vectors include Acyrthosiphon pisum (Harris), pea aphid, in Tasmania (Johnson and Guy 1986) and Macrosiphum euphorbiae (Thomas), potato aphid, in New Zealand (Wilson and Close 1973).

(Fig. 1)



SDV-D particles, X 100,000 (From Tamada 1975).

A. solani transmits SDV in a persistent manner. Minimum periods for the vector are acquisition access of 30 minutes, latent period of 12-21 hours, and inoculation access of 30 minutes (Damsteegt and Hewings 1987). The aphid can transmit SDV for up to 40 days (Kellock 1971, Tamada 1975) through aphid molts, but SDV is not known to multiply in the vector body. Nymphs transmit more efficiently than adults (Damsteegt and Hewings 1987, Tamada 1970, 1975, Tamada et al. 1969). Alates and apterae transmit equally well (Damsteegt and Snapp 1983). *A. solani* populations from different parts of the world differed in host preference and transmission efficiencies. A Japanese population of *A. solani* transmitted SDV more efficiently than other populations studied. California and New Zealand populations avoided soybeans but could transmit SDV to that crop (Damsteegt and Hewings 1986, 1987, Damsteegt et al. 1986).

After inoculation of soybeans, incubation ranged 6-13 days for SDV-D and 12-20 days for SDV-Y before symptoms appeared. Aphids recovered SDV in 48 to 72 hours from inoculated leaves and in 72-96 hours from uninoculated, symptomless leaves on the same plant at 20 °C (Tamada 1975).

Host range differences revealed two strains of SDV: dwarfing (SDV-D) and yellowing (SDV-Y). SDV-D infects *T. pratense* but not *P. vulgaris*, *Arachis hypogaea* (peanut), or *Lupinus cosenlini*; the reverse is true for SDV-Y (Tamada and Kojima 1977). Neither strain cross protects against infection by the other. Instead, a more severe infection results when both strains infect soybeans (Tamada 1973, 1975, 1977). Serological tests indicate that isolates from New Zealand and Tasmania represent a third strain (Damsteegt et al. 1986).

Characteristic Damage

Symptoms may vary depending on the host, the cultivar, plant age, the virus strain (Fig. 2), and other factors.

(Soybean)

Diseased plants mature later than healthy ones with leaflets remaining green at harvest (Banba et al. 1986, Tamada 1975).

SDV-D causes dwarfing (Fig. 3). Dwarfed plants show shortened petioles and internodes. Younger leaflets are faintly yellow. Older leaflets are dark green, smaller than normal, thick, brittle, and curl downward (Tamada 1973, 1975).

SDV-Y causes slightly cupping of very young leaves (Fig. 4). Older leaflet margins appear undulate (Fig. 5), instead of smooth (V. D. Damsteegt, pers. comm.). Leaflets become rugose or wrinkled, remain smaller than normal (Fig. 5), older leaflets become thickened and brittle; interveinal yellowing (Fig. 6) or marginal reddening appears on older leaves (Tamada

(Figs. 2-4)

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3

4

SDV-affected soybeans. 2. Plant habit on 'Shiro Tsurunoko,' left to right: SDV-Y, SDV-D, healthy. 3. Dwarfed plants of 'Wayne' with dark, curled leaves produced by SDV-D. 4. SDV-Y induced cupped leaves in 'Wayne' (2 from Tamada 1975; 3-4, courtesy V. D. Damsteegt).

1973, 1975). Stunting (Fig. 2) by SDV-Y is moderate compared to that by SDV-D. Plants affected by SDV-Y have a more open habit (Fig. 2) than healthy plants (V. D. Damsteegt, pers. comm.).

Plants infected by both virus strains show symptoms more severe than those infected with either strain alone, including very rugose leaves (Banba et al. 1986, Tamada 1973, 1975).

Younger soybeans are more susceptible than older soybeans. Unifoliolate and first-trifoliolate leaf stages are more

(Figs. 5-6)

5

6

SDV-Y induced symptoms on soybeans. 5. Rugose leaves with wavy margins in 'Wayne.' 6. Rugosity and interveinal yellowing, 4 weeks after inoculation (Courtesy V. D. Damsteegt).

susceptible than second-trifoliolate leaf stages and older (Damsteegt and Snapp 1983). Younger leaves are also more susceptible than older leaves (Tamada 1975).

(Subterranean clover)

Diseased plants exhibit leaves that turn bright red as they mature, with smaller leaves than those on healthy plants. Reddening begins at leaflet margins and gradually spreads interveinally towards the midrib. Later, leaf margins become necrotic. Plants are slightly stunted but continue to grow for a year (Wilson and Close 1973).

(Bean)

Plants are stunted. Leaves are yellowed, thickened, curl downward, and drop prematurely. Few pods are set (Ashby 1980).

(Broad bean)

Symptoms are more obvious in the cooler than the warmer part of the season. Leaves roll upwards about the midrib, interveinal chlorosis is a bright to dull yellow, and texture is thickened and rough (Johnstone 1978). Few pods are set (Ashby 1980).

(Pea)

Diseased plants are stunted with top yellowing. Plants become rigid and brittle with shoots proliferating from nodal buds at the plant base. Plants often succumb to secondary fungal root rot (Ashby 1980).

(Sugar beet) Older leaves exhibit interveinal chlorosis; leaves later turn bright yellow, sometimes with orange tints and veinal necrosis (Johnstone and Duffus 1984).

Detection Notes

Movement of the infected host plant, not the seed, from an infested country into new areas may introduce SDV. The viruliferous winged aphids can also introduce SDV by the vector hitchhiking on commodities to distant areas and feeding there on perennial hosts of SDV.

Check soybean fields for plants shorter than normal. Various leaflets may be smaller, curled downward, wrinkled; thick and brittle with younger leaflets yellow, and older leaflets dark green. Older leaflets may show interveinal yellowing.

In subterranean clover, look in late winter or early spring for the first symptoms. Leaves redden from the margins inwards. Plants may collapse and rot before the end of the growing season (Kellock 1971).

For identification, submit suspect fresh leaves labeled in double containers (one container inside another) with screw tops.

Biology and Etiology

Perennial legumes, red or white clovers, serve as winter hosts for SDV and the eggs of its aphid vector (Johnstone and McLean 1987, Tamada 1977). In Japan, _A. solani_ eggs hatch in early spring, and several aphid generations are produced. Eventually, the winged generation appears and acquires SDV through feeding on these infected legumes. Viruliferous alates fly to their secondary annual hosts (young soybeans) in late spring, feed, and thereby, introduce the virus into the plants (Tamada 1977). The infected plants [third leaflet stage] exhibit symptoms more than 6 weeks later (Banba et al. 1986). New aphids and soybeans become infected in turn as the aphids feed and reproduce for the rest of the season. Later in autumn, winged aphids develop, fly to perennial clovers, and lay overwintering eggs (Tamada 1977).

SDV is thus introduced from distant sources by alates and spread over a wide area (Tamada 1977). Spread in Japan occurs southward about 30 km per year. Rate of spread in a soybean field is more closely related to the number of alate migrants in the field than to the number of apterous aphids on the plants (Tamada 1975). In Tasmania, alates transmit SDV to subterranean clover seedlings in the autumn; apterae spread the virus in the autumn-sown crop in the spring (Johnstone and McLean 1987). One viruliferous aphid per soybean plant

transmitted SDV to 35 percent of the seedlings; 5-10 gave maximum infection (Damsteegt and Hewings 1987, Tamada 1970). Five apterae per subterranean clover plant transmitted to 20 percent of the plants (Wilson and Close 1973).

Controls

Resistant cultivars, if available, may be the most practical means of control. Other practices include scheduling sowing so young plants grow during periods of little aphid activity, avoiding planting near infected perennial hosts, and using biological and vector controls (Ashby and Johnstone 1985, Banba et al. 1986, Tamada 1975, 1977).

Difficulties have been experienced. In Japan, few resistant soybean cultivars are available. A crucial factor impeding eradication there is the widespread reservoir of SDV in wild, perennial clovers. In Tasmania, aphicides to control the vector in subterranean clover pastures were considered uneconomic (Johnstone and McLean 1987, Kellock 1971).

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